Case Report

Cortical Blindness in Eclampsia

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Abstract

Visual disturbances are common with severe preeclampsia, however, blindness either alone or with convulsions is uncommon. Most women with varying degree of amaurosis are found to have radiographic evidence of extensive occipital lobe hypodensities. In this case report, we present the clinical course of a woman in whom preeclampsia was further complicated by blindness. These were managed by standard pre eclampsia-eclampsia regimen used at our hospital which includes Diazepam/Phenytoin/Magnesium sulphate, therapy to control seizures, inj. labetolol to lower elevated blood pressure, intravenous fluid restriction and termination of pregnancy. Abnormal findings in these women were seen by magnetic resonance imaging technique. MRI demonstrated hyperdense lesion in both tempoparietal and occipital regions on T1W sequence. Total blindness persisted for 72 hrs and vision started improving and at the time of discharge vision improved significantly, however there was not much difference was seen on MRI.

Keywords: Eclampsia, Preeclampsia, Blindness, Magnetic resonance imaging, Cortical

Introduction

The humours Severe preeclampsia and eclampsia remains one of the leading causes of maternal and perinatal mortality and morbidity in many parts of the world. Patients may present with symptoms of headache and visual disturbances in the form of scotoma, sudden inability to focus, blurred vision and in severe cases to complete blindness. Although visual disturbances develop in perhaps 25% of women with severe pre eclampsia, blindness is rare and an incidence of 1-3% is reported with eclampsia¹. In past most cases of blindness were attributed to retinal abnormalities that include edema, vascular changes and detachment. Retinal detachment may cause altered vision although it is usually one sided and seldom causes total visual loss ². More recently case reports have emphasized cortical blindness which is characterized by intact pupillary light reflexes and normal ophthalmoscopic findings. Here we report the clinical course in a woman in whom preeclampsia-eclampsia was further

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complicated by blindness. Abnormal findings in these women were seen by magnetic resonance imaging techniques.

**Case Report**

24 yr old para 2 pregnant woman presented with headache, hypertension (190/130 mm of Hg) and generalized edema, seizures in the 38th week of pregnancy. Urine examination showed proteinuria 4+. Her serum creatinine was 1.0 mg/dl. Serum aspartate aminotransferase was 98.96 u /lt. She was treated with Inj. Labetelol to control her BP. Since she had good bishops score so labour was induced and augmented with oxytocin infusion. Soon after vaginal delivery she noted severe impairment of vision in both eyes. She was examined by an ophthalmologist and neurologist. MRI scan was advised which showed hyperdense lesion in both tempoparietal and occipital regions on T1W sequence. Cortical blindness lasted for 72 hrs and was followed by improvement of vision.

**Discussion**

The neuropathologic mechanism for eclampsia is unclear except when overt hemorrhage is identified. Some factors that have been implicated including cerebral vasospasm, hemorrhage, ischemia, edema as well as hypertensive and metabolic encephalopathy. Recently Schwartz et al proposed that the findings of preeclampsiaeclampsia can be explained by the loss of autoregulation of the posterior cerebral circulation. Aside from convulsions other dramatic neurologic effects, albeit uncommon, include blindness, an altered state of consciousness and coma. In the past most cases of blindness were attributed to retinal abnormalities that include edema, vascular changes and detachment. More recently case reports have emphasized cortical blindness, which is characterized by intact pupillary response and normal ophthalmoscopic findings. In 1980, Grimes et al reported the first case in which computed tomographic scanning was used to demonstrate reversible cortical lesion in a women with preeclampsia and temporary blindness. Others have reported low density areas predominantly in the occipital lobes of women with blindness associated with severe preeclampsia or eclampsia. Radiologic findings have ranged from normal to documentation of wide spread low density areas. The latter are nonenhancing and have been attributed to localized areas of decreased perfusion associated with arterial spasm, infarction or cerebral edema. Computed Tomography in our case with cortical blindness, showed occipital lobe hypodensities. Various authors have reported partial resolution of these radiologic hypodensities by 3 to 5 days with complete resolution within 14 days. We observed the improvement in vision but little change in MRI findings even after three weeks. There is other evidence that these lesions are induced by vascular changes. Using doppler velocimetry, Williams and Mclean showed that cerebral blood flow velocity is increased in pregnancy-induced hypertension, suggesting an increased resistance to flow. Velocity increased even more in the immediate puerperium. Thus the nature and duration of these reversible focal neurological lesions and transient cortical blindness are in concert with the characteristic reversible pathophysiologic changes of preeclampsiaeclampsia. The management guidelines are straightforward for women with severe preeclampsia or eclampsia in whom cortical blindness develops. Generally they are the same as for women without this sequela and include anticonvulsants, (Magnesium Sulfate, Phenytoin and Diazepam) for seizure prophylaxis, control of severe hypertension, and fluid restriction to avoid worsening of cerebral edema. Ophthalmologic and neurologic consultation along with neuroimaging is undertaken; however delivery should not be delayed unnecessarily. On the basis of previously published experiences with computed tomography in women with eclampsia, as well as the experience described here, we conclude that reversible cortical blindness.
associated with preeclampsia eclampsia may result from petechial hemorrhages and focal edema in the cerebral cortex.

**Ethical Approval:**
This case report was published after getting approval of the Ethics Committee of MMIMSR, Mullana (Ambala), India

**Conflict of Interest**
No Conflict of interest has been disclosed by the authors.

**References**